Freeform Search

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Term: l1 with l2

Display: 20 **Documents in Display Format:** - **Starting with Number** 1

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Search History

DATE: Tuesday, December 21, 2004 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT; PLUR=YES; OP=AND			
L5	l1 with l2	9	L5
L4	l1 near10 l2	9	L4
L3	l1 and L2	103	L3
L2	protect$ near7 against near6 (free adj radical or superoxide adj anion or heavy adj metal adj cation)	707	L2
L1	(neutraliz$ or eliminat$ or remov$) near7 (free adj radical or superoxide adj anion or heavy adj metal adj cation)	2016	L1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 9 of 9 returned.

☐ 1. 20030170199. 16 Dec 02. 11 Sep 03. Cosmetic and/or dermatological composition based on cocoa extracts. Leclere, Jacques. 424/74; 424/776 A61K007/06 A61K035/78.

☐ 2. 20020081288. 20 Jun 01. 27 Jun 02. Superoxide dismutase-4. Yu, Guo-Liang, et al. 424/94.4; 435/189 435/320.1 435/325 435/69.1 536/23.2 C12P021/02 C07H021/04 A61K038/44 C12N009/02.

☐ 3. 6635252. 20 Jun 01; 21 Oct 03. Antibodies to superoxide dismutase-4. Yu; Guo-Liang, et al. 424/146.1; 424/141.1 530/387.1 530/387.3 530/388.26 530/389.1. C07K016/00 C07K016/40 C07K016/46 A61K039/395.

☐ 4. 6433025. 13 Apr 00; 13 Aug 02. Method for retarding and preventing sunburn by UV light. Lorenz; R. Todd. 514/725; 424/400 424/401 424/59 514/691 514/724. A61K031/07 A61K031/045 A61K031/12 A61K009/00 A61K007/00 A61K007/42.

☐ 5. 6344214. 13 Dec 99; 05 Feb 02. Method for retarding and ameliorating fever blisters and canker sores. Lorenz; R. Todd. 424/451; 424/435 514/886 514/887 514/900 568/378. A61K035/70 A01N063/04.

☐ 6. 6258855. 08 Feb 00; 10 Jul 01. Method of retarding and ameliorating carpal tunnel syndrome. Lorenz; R. Todd, et al. 514/691;. A61K031/12.

☐ 7. 6194452. 30 Oct 98; 27 Feb 01. Stable pharmaceutical compositions including ascorbic acid and methods of using same. Murad; Howard. 514/474; 424/60. A61K031/34.

☐ 8. 5871729. 23 Jan 97; 16 Feb 99. Superoxide dismutase-4. Yu; Guo-Liang, et al. 424/94.4; 435/189. A61K038/44 C12N009/02.

☐ 9. 5506133. 11 Apr 94; 09 Apr 96. Superoxide dismutase-4. Yu; Gu-Liang, et al. 435/365; 435/252.3 435/254.11 435/320.1 536/23.2. C12N001/21 C12N005/10 C12N015/53 C12N015/63.

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Terms	Documents
L1 with L2	9

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h e b b g e e e f e e e e c che

08/907,041

=> d his

(FILE 'HOME' ENTERED AT 14:49:58 ON 21 DEC 2004)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 14:50:09 ON 21 DEC 2004

L1 2025 S (NEUTRALIZ? OR ELIMINAT? OR REMOV?)(7A)(FREE(W)RADICAL OR SUP
L2 3138 S PROTECT?(7A)AGAINST(6A)(FREE(W)RADICAL OR SUPEROXIDE(W)ANION
L3 15 S L1(S)L2
L4 10 DUP REM L3 (5 DUPLICATES REMOVED)

=> d au ti so pi ab 1-10 l4

L4 ANSWER 1 OF 10 MEDLINE on STN DUPLICATE 1
AU Le Bourg Eric; Fournier Didier
TI Is lifespan extension accompanied by improved antioxidant defences? A study of superoxide dismutase and catalase in Drosophila melanogaster flies that lived in hypergravity at a young age.
SO Biogerontology, (2004) 5 (4) 261-6.
Journal code: 100930043. ISSN: 1389-5729.
AB It has been previously shown that exposing Drosophila melanogaster flies to hypergravity (3g or 5g) at a young age for 2 weeks increases male longevity, resistance to heat in both sexes, and delays behavioural ageing, but the causes of these effects are unknown. We hypothesised that these flies could be well **protected against** **free radical** attacks and, if this **protection** persists after **removal** from hypergravity, can better resist **free radicals** and finally live longer than flies that have always lived at 1g. If so, the activity of enzymes detoxifying free radicals superoxide dismutase and catalase should be increased in flies that have lived in hypergravity. Results showed that no effect of hypergravity on the activity of these enzymes was observed at 2, 4 or 6 weeks of age. The greater longevity of male flies that have lived in hypergravity at a young age thus cannot be explained by the activity changes of these major antioxidant enzymes.

L4 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
AU Wei, Anyang; Zhou, Chunlan
TI Effects of trace element selenium in the defense against free radical damage
SO Guangdong Weiliang Yuansu Kexue (2001), 8(5), 23-25
CODEN: GWYKF3; ISSN: 1006-446X
AB A review with 10 refs. on the **protective** action of trace element Se **against free radical** damages, by **elimination** of **free radicals** and the interruption of lipid peroxidn. via the action of glutathione peroxidase. The roles of Se as an active constituent of glutathione peroxidase in suppression of lipid peroxidn. and maintenance of cellular integrity are also discussed.

L4 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
AU Qi, Kezong; Wang, Linan; Shu, Yuanshan
TI Influences of united-infusion of cold and warm blood cardioplegia on oxygen free radical metabolism in the canine
SO Zhongguo Shouyi Xuebao (1999), 19(1), 76-79
CODEN: ZSXUF5; ISSN: 1005-4545
AB To investigate the effects of blood cardioplegia on myocardial protection, twenty-eight mongrel dogs were equally divided into four groups:group A, infusion with cold crystalloid (4°); group B, infusion with cold blood cardioplegia (10°); group C, on the basis of group A, infusion with warm blood cardioplegia (37°) at 5 min before opening aorta clamping; group D, infusion with cold blood cardioplegia (20°), combined with warm blood cardioplegia the same as group C.

Undergoing same CPB, the coronary venous blood samples were collected during ischemia and reperfusion, to measure the contents of serum malondialdehyde (MDA), serum superoxide dismutase (SOD); glutathione peroxidase (GSH-Px) activities. The results showed: the serum MDA levels of all group increased, activities of serum SOD and GSH-Px decreased following ischemia and reperfusion. In group A, the contents of MDA increased dramatically, activities of SOD, GSH-Px decreased obviously following reperfusion as compared with group D ($P<0.01$). The results suggested that the united-infusion of cold and warm blood cardioplegia may **eliminate free radical** products, and thereby effectively **protect** the myocardial cells **against** injury of ischemia and reperfusion.

L4 ANSWER 4 OF 10 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AU Boldyrev A A (Reprint); Stvolinsky S L; Tyulina O V; Koshelev V B; Hori N; Carpenter D O

TI Biochemical and physiological evidence that carnosine is an endogenous neuroprotector against free radicals

SO CELLULAR AND MOLECULAR NEUROBIOLOGY, (APR 1997) Vol. 17, No. 2, pp. 259-271.
Publisher: PLENUM PUBL CORP, 233 SPRING ST, NEW YORK, NY 10013.
ISSN: 0272-4340.

AB 1. Carnosine, anserine, and homocarnosine are endogenous dipeptides concentrated in brain and muscle whose biological functions remain in doubt.

2. We have tested the hypothesis that these compounds function as endogenous protective substances against molecular and cellular damage from free radicals, using two isolated enzyme systems and two models of ischemic brain injury. Carnosine and homocarnosine are both effective in activating brain Na, K-ATPase measured under optimal conditions and in reducing the loss of its activity caused by incubation with hydrogen peroxide.

3. In contrast, all three endogenous dipeptides cause a reduction in the activity of brain tyrosine hydroxylase, an enzyme activated by free radicals, In hippocampal brain slices subjected to ischemia, carnosine increased the time to loss of excitability.

4. In in vivo experiments on rats under experimental hypobaric hypoxia, carnosine increased the time to loss of ability to stand and breath and decreased the time to recovery.

5. These actions are explicable by effects of carnosine and related compounds which **neutralize free radicals**, particularly hydroxyl radicals. In all experiments the effective concentration of carnosine was comparable to or lower than those found in brain. These observations provide further support for the conclusion that **protection against free radical** damage is a major role of carnosine, anserine, and homocarnosine.

L4 ANSWER 5 OF 10 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AU Lee E H (Reprint); Upadhyaya A; Agrawal M; Rowland R A

TI Mechanisms of ethylenediurea (EDU) induced ozone protection: Reexamination of free radical scavenger systems in snap bean exposed to O-3

SO ENVIRONMENTAL AND EXPERIMENTAL BOTANY, (NOV 1997) Vol. 38, No. 2, pp. 199-209.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD, ENGLAND OX5 1GB.
ISSN: 0098-8472.

AB Ethylenediurea (EDU), N-[2-(2-oxo-1-imidazolidinyl) ethyl]-N'-phenylurea is known to prevent ozone (O-3) damage to leaf tissues. However, the mechanisms of protection are unclear. We tested the hypothesis that EDU **protects against** O-3 damage by scavenging hydroxyl **free radicals** ((OH)-O-.). An in vitro study involving the use of high-performance liquid chromatography

equipped with an electrochemical detector (HPLC-EC) showed that EDU does not serve as an antioxidant to **remove** .OH **free radicals**. Effects of O-3 and EDU (soil drench) on leaf antioxidant scavenger systems (AOSS) were also studied. The first fully expanded trifoliate leaves of O-3-sensitive snap bean (Phaseolus vulgaris cv. Bush Blue Lake 290) was examined. Measurements were made before and after a single O-3 exposure (0.30 mu l l(-1) O-3 for 3 h). Pretreatment with EDU 48 h before exposure protected against O-3-induced necrosis and chlorosis. EDU pretreatments did not alter superoxide dismutase (SOD), guaiacol-peroxidase (GPX), ascorbate peroxidase (APX) and glutathione reductase (GR) activities. However, O-3-fumigated plants (no EDU) showed elevated SOD activity with decreased GR activity. EDU-treated plants exposed to O-3 stress showed no measurable loss of GR activity. These tissues maintained high levels of total glutathione [i.e. reduced glutathione (GSH) + oxidized glutathione (GSSG)] contents, and had higher GSH/GSSG ratios than the controls at the end of 3 h exposure to O-3. These data suggest that EDU protection against O-3 damage in plants do not necessarily involve the direct stimulation or induction of antioxidative enzyme defense mechanisms. Instead, protection may result from a more general retention of chlorophyll and maintenance of GR and GSH levels during O-3 exposure. (C) 1997 Elsevier Science B.V.

L4 ANSWER 6 OF 10 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AU REITER R J (Reprint); MELCHIORRI D; SEWERYNEK E; POEGGELER B; BARLOWWALDEN L; CHUANG J I; ORTIZ G G; ACUNACASTROVIEJO D

TI A REVIEW OF THE EVIDENCE SUPPORTING MELATONINS ROLE AS AN ANTIOXIDANT

SO JOURNAL OF PINEAL RESEARCH, (JAN 1995) Vol. 18, No. 1, pp. 1-11. ISSN: 0742-3098.

AB This survey summarizes the findings, accumulated within the last 2 years, concerning melatonin's role in defending against toxic free radicals. Free radicals are chemical constituents that have an unpaired electron in their outer orbital and, because of this feature, are highly reactive. Inspired oxygen, which sustains life, also is harmful because up to 5% of the oxygen (O2) taken in is converted to oxygen-free radicals. The addition of a single electron to O-2 produces the superoxide anion radical (O-2 radical anion); O2 radical anion is catalytic-reduced by superoxide dismutase, to hydrogen peroxide (H2O2) Although H2O2 is not itself a free radical, it can be toxic at high concentrations and, more importantly, it can be reduced to the hydroxyl radical (. OH). The . OH is the most toxic of the oxygen-based radical cals and it wreaks havoc within cells, particularly with macromolecules. In recent in vitro studies, melatonin was shown to be a very efficient neutralizer of the . OH; indeed, in the system used to test its free radical scavenging ability it was found to be significantly more effective than the well known antioxidant, glutathione (GSH), in doing so. Likewise, melatonin has been shown to stimulate glutathione peroxidase (GSH-Px) activity in neural tissue; GSH-PX metabolizes reduced glutathione to its oxidized form and in doing so it converts H2O2 to H2O, thereby reducing generation of the . OH by eliminating its precursor. More recent studies have shown that melatonin is also a more efficient scavenger of the peroxyl radical than is vitamin E. The peroxyl radical is generated during lipid peroxidation and propagates the chain reaction that leads to massive lipid destruction in cell membranes. In vivo studies have demonstrated that melatonin is remarkably potent in **protecting against free radical** damage induced by a variety of means. Thus, DNA damage resulting from either the exposure of animals to the chemical carcinogen safrole or to ionizing radiation is markedly reduced when melatonin is co-administered. Likewise, the induction of cataracts, generally accepted as being a consequence of free radical attack on lenticular macromolecules, in newborn rats injected with a GSH-depleting drug are prevented when the animals are given daily melatonin injections. Also, paraquat-induced lipid peroxidation in the lungs of rats is overcome when they also receive melatonin during the exposure period. Paraquat is a

highly toxic herbicide that inflicts at least part of its damage by generating free radicals. Finally, bacterial endotoxin (lipopolysaccharide or LPS)-induced free radical damage to a variety of organs is highly significantly reduced when melatonin is also administered; LPS, like paraquat, produces at least part of its damage to cells by inducing the formation of free radicals. Physiological melatonin concentrations have also been shown to inhibit the nitric oxide (NO .)-generating enzyme, nitric oxide synthase. The reduction of NO . production would contribute to melatonin's antioxidant action since NO . can generate the peroxynitrite anion, which can degrade into the . OH. Thus, melatonin seems to have multiple ways either to reduce **free radical** generation or, once produced, to **neutralize** them. Melatonin accomplishes these actions without membrane receptors, indicating that the indole has important metabolic functions in every cell in the organism, not only those that obviously contain membrane receptors for this molecule.

L4 ANSWER 7 OF 10 MEDLINE on STN DUPLICATE 2
AU Tsuchida T; Yasuyama T; Higuchi K; Watanabe A; Urakami T; Akaike T; Sato K; Maeda H
TI The protective effect of pyrroloquinoline quinone and its derivatives against carbon tetrachloride-induced liver injury of rats.
SO Journal of gastroenterology and hepatology, (1993 Jul-Aug) 8 (4) 342-7. Journal code: 8607909. ISSN: 0815-9319.
AB Pyrroloquinoline quinone (PQQ) and its derivative, oxazo pyrroloquinoline (OPQ-G), protected rats from experimental liver injury induced by carbon tetrachloride (CCl4) in vivo. This effect was observed after an intraperitoneal injection of 5 mg/kg PQQ or OPQ-G, which was given twice, 10 min and 1 h before CCl4 administration. Pyrroloquinoline quinone protected primary cultured rat hepatocytes from CCl4 toxicity in vitro. This protection was most effective at a concentration of 3 mumol/L PQQ. Pyrroloquinoline quinone derivatives (oxazo pyrroloquinoline, methyl-thioethyl oxazo pyrroloquinoline and PQQ-allylester) also protected the hepatocytes from CCl4 toxicity. Pyrroloquinoline quinone and its derivatives inhibited the lucigenin-enhanced chemiluminescence from isolated hepatocytes initiated by CCl4. These results suggest that **eliminating free radicals** is one of the **protective** mechanisms of PQQ and its derivatives **against** CCl4-induced liver injury.

L4 ANSWER 8 OF 10 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU ALTORJAY I (Reprint); DALMI L; SARI B; IMRE S; BALLA G
TI THE EFFECT OF SILIBININ (LEGALON(R)) ON THE FREE-RADICAL SCAVENGER MECHANISMS OF HUMAN ERYTHROCYTES IN-VITRO
SO ACTA PHYSIOLOGICA HUNGARICA, (1992) Vol. 80, No. 1-4, pp. 375-380. ISSN: 0231-424X.
AB The effect of LegalonR was investigated parallel with that of AdriblastinaR (doxorubicin) and paracetamol on some parameters characterizing the free radical scavenger mechanisms of human erythrocytes in vitro and on the time of acid haemolysis performed in aggregometer. Observations suggest that Adriblastina enhances the lipid peroxidation of the membrane of red blood cells, while paracetamol causes significant depletion of intracellular glutathione level, thus decreasing the **free radical eliminating** capacity of the glutathione peroxidase system. LegalonR on the other hand, is able to increase the activity of both superoxide dismutase and glutathione peroxidase, which may explain the **protective** effect of the drug **against free radicals** and also the stabilizing effect on the red blood cell membrane, shown by the increase of the time of full haemolysis.

L4 ANSWER 9 OF 10 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AU BOLTER C J [Reprint author]; CHEFURKA W
TI THE EFFECT OF PHOSPHINE TREATMENT ON SUPEROXIDE DISMUTASE CATALASE AND PEROXIDASE IN THE GRANARY WEEVIL SITOPHILUS-GRANARIUS.
SO Pesticide Biochemistry and Physiology, (1990) Vol. 36, No. 1, pp. 52-60. CODEN: PCBPBS. ISSN: 0048-3575.
AB Previous studies have shown that the fumigant insecticide phosphine (PH3) inhibits cytochrome c oxidase and that a direct relationship exists between oxygen concentration during fumigation and insect mortality. Recently, it was shown that PH3 stimulated the release of hydrogen peroxide (H2O2) from isolated insect mitochondria in vitro and it was hypothesized that treatment with PH3 in vivo could result in the generation of superoxide radicals (O2-) by the inhibited electron transport chain. The cell contains a complex oxygen defense system to **protect** itself **against** oxygen-derived **free radicals**, including three enzymes: superoxide dismutase (SOD), which **removes** O2-, the catalase (CAT), and peroxidase (PER), which remove H2O2. The effect of PH3 treatment on this antioxidant enzyme system was investigated using PH3-susceptible (S) and -resistant (R) granary weevils. No glutathione peroxidase activity was found in this species. However, it did contain peroxidase activity that was observed using p-phenylenediamine as an indicator. Peroxidase activity was the same in S- and R-insects and was reduced by 65% in S- and 45% in R-insects 3 days after treatment (LD30). Catalase activity was significantly higher (62%) in S-insects than R. This activity was inhibited by 34% in S-insects 3 days after treatment (LD30), but was unaffected in R-insects. A pyrogallol assay was used to measure superoxide dismutase. Two isozymes were present, a cyanide (CN)-insensitive form in the mitochondria and a CN-sensitive form in the cytosol. Activity of the latter enzyme increased twofold after in vivo PH3 treatment (LD30) in S-insects, while no change was observed in R-insects. This study demonstrates that PH3 treatment has a significant effect on the enzymes involved in oxygen defense. Elevated SOD activity probably occurred in response to an increase in O2- generation and this coupled with a reduction in both CAT and PER activity could result in an accumulation of H2O2 and the consequent production of the hydroxyl radical (HO.), a powerful oxidizing agent. These results indicate that insect mortality could be attributed to accumulation by oxygen-derived free radicals which eventually destroy the cell integrity.

L4 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN
AU Przybyszewski, Waldemar M.; Malec, Janina
TI Protection against hydroxyurea-induced cytotoxic effects in L5178Y cells by free radical scavengers
SO Cancer Letters (Shannon, Ireland) (1982), 17(2), 223-8 CODEN: CALEDQ; ISSN: 0304-3835
AB Exposure of L5178Y cells in culture to 1 mM hydroxyurea (HU) [127-07-1] for 3 h followed by 24 h incubation in an HU-free medium induced an abnormal enlargement of about 40% of the cells in the population and post-treatment reduction of DNA synthesis in comparison with control cells. These effects were used to examine the protection afforded by free radical scavengers against HU-induced cytotoxicity. With careful choice of conditions (suitable concentration of the protective agent, pretreatment of cells) substantial protective effect of α-tocopherol acetate [58-95-7], sodium benzoate [532-32-1], acetylsalicylic acid [50-78-2], catalase [9001-05-2], peroxidase [9003-99-0], or superoxide dismutase [9054-89-1] can be achieved.

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     (FILE 'HOME' ENTERED AT 14:49:58 ON 21 DEC 2004)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 14:50:09 ON 21 DEC
     2004
L1          2025 S (NEUTRALIZ? OR ELIMINAT? OR REMOV?)(7A)(FREE(W)RADICAL OR SUP
L2          3138 S PROTECT?(7A)AGAINST(6A)(FREE(W)RADICAL OR SUPEROXIDE(W)ANION
L3            15 S L1(S)L2
L4            10 DUP REM L3 (5 DUPLICATES REMOVED)
L5        474926 S (POLYNUCLEOTIDE OR CDNA OR DNA OR NUCLEIC(W)ACID)(7A)(PROTEIN
L6            10 S (POLYNUCLEOTIDE OR CDNA OR DNA OR NUCLEIC(W)ACID)(7A)(PROTEIN
L7            24 S (POLYNUCLEOTIDE OR CDNA OR DNA OR NUCLEIC(W)ACID)(7A)(GLUTAMY
L8             0 S L2 AND L6
L9             0 S L2 AND L7
L10            4 S L1 AND L6
L11            0 S L1 AND L7
L12            0 S L4 AND L5
L13           46 S L1 AND L5
L14           16 S L1(S)L5
L15            2 DUP REM L10 (2 DUPLICATES REMOVED)
L16           12 DUP REM L14 (4 DUPLICATES REMOVED)

=> d au ti so pi ab 1-2 l15

L15  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2004 ACS on STN
AU   Zwier, Henk J.; Le Poole, Rik A. C.
TI   Antioxidants against oxidative stress in obesity and diabetes
SO   NutraCos (2003), 2(5), 17-20
     CODEN: NUTRCP; ISSN: 1720-4011
AB   A review.  The worldwide increase in the prevalence of obesity in affluent
     societies is alarming.  As obesity is a major risk factor for the
     development of diabetes type 2 also the incidence of diabetes type 2 rises
     rapidly.  Obese people can have very high levels of oxidative stress which
     is a direct consequence of high blood levels of glucose (hyperglycemia).
     Therefore oxidative stress seems to be the "connector" between obesity and
     its complications like diabetes type 2 and heart diseases.  The build-up
     of reactive oxygen species (ROS) occurs in normal metabolic processes
     through the production of free radicals', unstable, elec. charged oxygen mols.
     in the quest to find a "mate" and become stable, free
     radicals interact with the nearest mol. targeting proteins
     , fatty acids or DNA if not neutralized rapidly by
     antioxidants systems, the free radicals may create
     more bee radicals or cause damage to cell membranes, blood vessel walls,
     lipoproteins, and even the nucleus (DNA) of the cell.  These processes can
     lead to cell death.  That obesity and diabetes are associated with an
     increased oxidative stress has prompted interest in the use of antioxidant
     supplements including the vitamins C and E, carotenoids α-lipoic
     acid and the several flavonoids.  In vivo antioxidants have been shown to
     be able to counteract hyperglycemia-induced oxidative alterations.  As
     antioxidant work closely together, a mixture of dietary antioxidants
     including the vitamin E and C, α-lipoic acid, carotenoids and
     polyphenols including flavonoids, might possess the best option in
     enhancing the qualify of life of obese and diabetic individuals and would
     help strengthen the body's natural defenses.  Of course, antioxidants will
     also provide protection against oxidative stress as induced by
     environmental factors such as air pollution by cigarette smoke and
     combusting engines and UV light.

L15  ANSWER 2 OF 2     MEDLINE on STN                    DUPLICATE 1
AU   Larsen C J
TI   [The BCL2 gene, prototype of a gene family that controls programmed cell
     death (apoptosis)].
     Le gene BCL2, chef de file d'une famille de genes controlant la mort
```

cellulaire programmee (apoptose).
SO Annales de genetique, (1994) 37 (3) 121-34. Ref: 99
Journal code: 0370562. ISSN: 0003-3995.
AB The BCL2 gene is the most representative member of a family of genes that control cell homeostatic processes in the course of the developmental and adult life. Some members of the BCL2 family (bcl-2 alpha, bcl-xL) inhibit apoptosis, whereas some other (Bax, Bclxs) induce it. The biological activity of these proteins is dictated by: 1) their capacity to be integrated in specific membranes of the cytoplasm; 2) their ability to homo- or hetero-dimerize, due to the presence of two highly conserved domains which are a signature of this gene family. The bcl-2 protein exhibits two main biochemical properties: it acts in an antioxidant metabolic pathway aimed at **eliminating** oxygene **free radicals** that induce lesions in **DNA**, lipids and **proteins**; it modulates intracellular Ca++ fluxes. BCL2 (and presumably its congeners) interplay with other genes involved in the tight control of cell proliferation and programmed cell death (c-myc, p53). A more comprehensive view of BCL2 functions should benefit to cancer chemotherapy by improving rational approach of the antitumor drug mechanisms.

=> d au ti so pi 1-12 l16

L16 ANSWER 1 OF 12 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Meseguer M (Reprint); Garrido N; Simon C; Pellicer A; Remohi J
TI Concentration of glutathione and expression of glutathione peroxidases 1 and 4 in fresh sperm provide a forecast of the outcome of cryopreservation of human spermatozoa
SO JOURNAL OF ANDROLOGY, (SEP-OCT 2004) Vol. 25, No. 5, pp. 773-780.
Publisher: AMER SOC ANDROLOGY, INC, C/O ALLEN PRESS, INC PO BOX 368, LAWRENCE, KS 66044 USA.
ISSN: 0196-3635.

L16 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN
AU Zwier, Henk J.; Le Poole, Rik A. C.
TI Antioxidants against oxidative stress in obesity and diabetes
SO NutraCos (2003), 2(5), 17-20
CODEN: NUTRCP; ISSN: 1720-4011

L16 ANSWER 3 OF 12 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Inamdar K V; Yu Y; Povirk L F (Reprint)
TI Resistance of 3 '-phosphoglycolate DNA ends to digestion by mammalian DNase III
SO RADIATION RESEARCH, (MAR 2002) Vol. 157, No. 3, pp. 306-311.
Publisher: RADIATION RESEARCH SOC, 820 JORIE BOULEVARD, OAK BROOK, IL 60523 USA.
ISSN: 0033-7587.

L16 ANSWER 4 OF 12 MEDLINE on STN DUPLICATE 1
AU Christen Y
TI Oxidative stress and Alzheimer disease.
SO American journal of clinical nutrition, (2000 Feb) 71 (2) 621S-629S. Ref: 117
Journal code: 0376027. ISSN: 0002-9165.

L16 ANSWER 5 OF 12 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Felzenszwalb I (Reprint); deMattos J C P; Bernardo M; CaldeiradeAraujo A
TI Shark cartilage-containing preparation: Protection against reactive oxygen species
SO FOOD AND CHEMICAL TOXICOLOGY, (DEC 1998) Vol. 36, No. 12, pp. 1079-&.

Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.
ISSN: 0278-6915.

L16 ANSWER 6 OF 12 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Wilson J X (Reprint)
TI Antioxidant defense of the brain: a role for astrocytes
SO CANADIAN JOURNAL OF PHYSIOLOGY AND PHARMACOLOGY, (OCT-NOV 1997) Vol. 75, No. 10-11, pp. 1149-1163.
Publisher: NATL RESEARCH COUNCIL CANADA, RESEARCH JOURNALS, MONTREAL RD, OTTAWA ON K1A 0R6, CANADA.
ISSN: 0008-4212.

L16 ANSWER 7 OF 12 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Bains J S (Reprint); Kakkar R; Sharma S P
TI Increased longevity, reduced fecundity, and delayed development in fruitfly (Zaprionus paravittiger) fed on butylated hydroxy anisole
SO PROCEEDINGS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE, (JUL 1997) Vol. 215, No. 3, pp. 237-242.
Publisher: BLACKWELL SCIENCE INC, 350 MAIN ST, MALDEN, MA 02148.
ISSN: 0037-9727.

L16 ANSWER 8 OF 12 MEDLINE on STN DUPLICATE 2
AU Larsen C J
TI [The BCL2 gene, prototype of a gene family that controls programmed cell death (apoptosis)].
Le gene BCL2, chef de file d'une famille de genes controlant la mort cellulaire programmee (apoptose).
SO Annales de genetique, (1994) 37 (3) 121-34. Ref: 99
Journal code: 0370562. ISSN: 0003-3995.

L16 ANSWER 9 OF 12 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU SATOH M S (Reprint); JONES C J; WOOD R D; LINDAHL T
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08/907,041

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Inventor Name Search Result

Your Search was:

Last Name = GREENBERGER
First Name = JOEL S.

Application#	Patent#	Status	Date Filed	Title	Inventor Name 24
09292056	Not Issued	120	04/14/1999	METHOD AND APPARATUS FOR HOLDING CELLS	GREENBERGER, JOEL S.
09224048	6387366	150	12/31/1998	METHODS FOR REDUCING ADVERSE SIDE EFFECTS ASSOCIATED WITH CELLULAR TRANSPLANTATION	GREENBERGER, JOEL S.
09222172	6270472	150	12/29/1998	APPARATUS AND A METHOD FOR AUTOMATICALLY INTRODUCING IMPLANTS INTO SOFT TISSUE WITH ADJUSTABLE SPACING	GREENBERGER, JOEL S.
09107051	Not Issued	161	06/30/1998	METHODS OF PREPARING BONE MARROW STROMAL CELLS FOR USE IN GENE THERAPY	GREENBERGER, JOEL S.
09094918	5962323	150	06/15/1998	EXPANSION OF BONE MARROW STROMAL CELLS	GREENBERGER, JOEL S.
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08914631	5993801	150	08/19/1997	GENE THERAPY USING STROMAL CELLS	GREENBERGER, JOEL S.
08907041	Not Issued	071	08/06/1997	PROTECTION FROM IONIZING IRRADIATION OR CHEMOTHERAPEUTIC DRUG DAMAGE BY IN VIVO GENE THERAPY	GREENBERGER, JOEL S.
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08602145	6025336	150	02/15/1996	DETERMINING EXPOSURE TO IONIZING RADIATION AGENT WITH PERSISTENT BIOLOGICAL MARKERS	GREENBERGER , JOEL S.
08581059	5766950	150	12/29/1995	EXPANSION OF BONE MARROW STROMAL CELLS	GREENBERGER , JOEL S.
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